**The information provided here is for informational purposes only and does not constitute medical advice. Always consult with a qualified healthcare professional for any health concerns or before making any decisions related to your health or treatment.**

**If you suspect someone has overdosed on GHB, seek immediate medical attention by calling emergency services or going to the nearest hospital. Do not attempt to treat the person yourself.**

## Recovery Kernel:

* **Administer Naloxone if available and trained to do so.**

* Monitor vital signs such as breathing, heart rate, and blood pressure.
* Provide supportive care such as keeping the person calm and comfortable.
* Transport the person to a medical facility for further evaluation and treatment.

**Naloxone:**

* Naloxone is an opioid antagonist that can reverse the effects of an overdose.
* It is available in various forms, including nasal spray and injection.
* Follow the instructions provided by your healthcare provider or emergency services personnel on how to administer Naloxone.

**COMT:**

* **COMT is an enzyme involved in the metabolism of certain medications, including some used to treat GHB overdose.**
* **Your doctor may consider adjusting the dosage of these medications based on your individual COMT levels.**

**DRD2:**

* **DRD2 is a dopamine receptor that plays a role in the effects of GHB on the brain.**
* **Research suggests that individuals with certain variations in the DRD2 gene may be more susceptible to the addictive properties of GHB.**

**MTHFR:**

* **MTHFR is a gene involved in folate metabolism, which is**

important for overall health and well-being.

* Some studies suggest a possible link between MTHFR gene variations and substance use disorders, but further research is needed.

Blueprint:

* A comprehensive plan for recovery from GHB addiction should include various components such as medical detoxification, behavioral therapy, support groups, and aftercare programs.
* It is essential to work with a qualified healthcare professional or addiction specialist to develop a personalized recovery plan that addresses your specific needs and goals.

# findahelpline.com:
# * This website provides information and resources for individuals seeking help with substance abuse and mental health issues.
# * You can find local support groups, treatment centers, and other helpful services through this platform.

### Using the Recovery Kernel Framework.

GHB · GBL · 1,4-BD · Sodium Oxybate (Xyrem) · Liquid Ecstasy · G · Fantasy.

**⚠ RESURSOS PARA REDUCCIÓN DE DAÑOS — NO ES UN CONSEJO MÉDICO | Crisis: llama al 000 o envía un mensaje de texto al 13 11 14.**

**GHB withdrawal can cause fatal seizures and psychosis.**

**DO NOT STOP ABRUPTLY WITHOUT MEDICAL SUPERVISION IF DOSING EVERY FEW HOURS.**

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## Een bericht voordat u begint.

GHB recovery may be the least documented and least supported recovery journey in this entire series. It is not because GHB use disorder is rare — it is because GHB exists in a strange cultural and clinical blind spot. It is simultaneously a Schedule I controlled substance, an FDA-approved medication (sodium oxybate / Xyrem, for narcolepsy), a harm reduction tool used by some to manage alcohol cravings, a chemsex drug prevalent in LGBTQ+ nightlife cultures, and a molecule the human brain produces naturally in small amounts. Its users span a broader range of contexts than almost any other substance in Deze gidsenreeks behandelt de volgende situaties: een persoon die kampt met narcolepsie, een frequente

gebruiker van "The Weekend Club" die merkte dat hij/zij het steeds vaker nodig had (elke paar uur), en iemand die deelneemt aan chemsex en wiens recreatief gebruik is uitgegroeid tot dagelijks gebruik.

What unites all of these stories is the biology: GHB acts primarily as a GABA-B agonist and also activates its own GHB-specific receptors, producing a GABA-dependent physical dependence that — when severe — is as medically dangerous as any substance known. The GABRA2 high-risk variant that the compendium identifies as the 'Hard Fail' genotype for combining alcohol with GABAergic agents applies here with full force. GHB withdrawal at high frequency and doses is a medical emergency with a documented mortality risk.

This guide covers the full spectrum — from recreational GHB users who have developed problematic patterns, to people dosing every two to three hours around the clock, to those prescribed sodium oxybate who need to reduce or discontinue. The biology is the same. The medical urgency varies with the frequency and dose.

Read Section 0 first. Then continue.

# Section 0 — Immediate Safety: Read This First

**⚠ Abstinência de GHB em uso frequente (a cada 2 a 4 horas) pode causar convulsões, delírio e morte dentro de 2 a 6 horas da última dose. Isso não é exagero. Não interrompa o uso abruptamente.**

## Chi ha bisogno di disintossicazione medica? La valutazione.

**THE CENTURION — The line that determines whether you continue reading or call a doctor first.**

Okay, I understand. I will answer truthfully.

| Question | If yes: |
|---|---|
| Are you currently taking GHB/GBL/1,4-BD more than 3 times a day? | Detoxificación médica probablemente necesaria. Consulte a su médico o acuda a un centro de atención urgente antes de reducir la dosis. |
| Have you experienced shaking, sweating, or extreme anxiety when you missed a dose? | Physical dependence is present. Do not stop abruptly. Medical supervision required. |
| Have you ever experienced seizures, confusion, or hallucinations when stopping GHB use? | Grave dependencia. Se requiere una desintoxicación médica de emergencia; no intente esto solo. |
| Are you taking the medication every 2 hours, or more often, to avoid withdrawal symptoms? | Alta gravidade de dependência. A desintoxicação supervisionada por médicos em ambiente hospitalar é o primeiro passo apropriado. |
| Are you also regularly using alcohol or benzodiazepines alongside GHB? | Combined GABAergic withdrawal is significantly more dangerous. Medical supervision is essential. |

If you answered no to all of the above — you dose once or twice daily, have not experienced physical withdrawal symptoms when missing a dose, and have a stable pattern — a carefully monitored taper may be possible with medical guidance but without inpatient detox. Discuss with a physician before reducing.

## GHB Overdose Recognition and Response

### Recognising a GHB overdose:

* Severe respiratory depression (slowed or stopped breathing)
* Loss of consciousness
* Seizures
* Vomiting
* Blue lips or fingertips

### Responding to a GHB overdose:

1. Call for medical help immediately. Do not hesitate. Medical help is not failure.
2. Place the person in the recovery position.
3. If the person is vomiting, turn them on their side to prevent choking.
4. Administer Naloxone if available and trained to do so.
5. Stay with the person until medical help arrives.
6. Provide information about the substance taken to paramedics.

### Important considerations:

* GHB can cause rapid and severe respiratory depression, which can be life-threatening.
* It is important to recognise the signs of a GHB overdose quickly and seek immediate medical attention.
* Naloxone may not reverse the effects of GHB, but it can help to prevent or treat opioid overdose if GHB is mixed with opioids.
* Do not leave the person unattended.
* Provide as much information as possible to paramedics about the substance taken and the amount taken.

### Additional resources:

* findahelpline.com

**⚠ OVERDOSE DUE TO GHB: SEDATION → UNCONSCIOUSNESS → RESPIRATORY DEPRESSION → DEATH. THE TIMEFRAME IS VERY SHORT. THE AMOUNT THAT CAUSES SEDATION AND THE AMOUNT THAT CAN BE FATAL ARE VERY CLOSE.**

- Unresponsive — unable to be roused by voice, sternal rub, or pain stimulus.
- Lenta, superficial o ausente respiração — a principal causa de morte por GHB.
- Lèvres ou ongles bleus ou gris : manque d'oxygène.
- Vomiting while unconscious — aspiration risk; roll to recovery position immediately.
- Seizure — a medical emergency at any stage.

**⚠ THERE IS NO REVERSAL AGENT FOR GHB OVERDOSE. NALOXONE DOES NOT WORK ON GHB.**

Unlike opioid overdose, there is no pharmacological reversal for GHB overdose. Supportive care — airway management, oxygen, monitoring — is the treatment. This is why calling 000 immediately is the only appropriate response to a suspected GHB overdose. Do not wait to see if the person wakes up. GHB overdose can progress from sedation to respiratory arrest in minutes.

**GHB + alcohol combination: The compendium's 'Hard Fail' scenario for GABRA2 high-risk individuals. Both substances are GABAergic. Their combined effect is not additive — it is synergistic and can produce respiratory depression and death at doses that would be non-fatal individually. This combination kills. Do not combine.**

**GHB + any other depressant: The combination of GHB with benzodiazepines, opioids, ketamine, alcohol, or antihistamines significantly increases the risk of overdose and death. There are no exceptions.**

## The Dose Measurement Problem — The Most Unique Danger of GHB.

### THE CENTURION — GHB wordt verkocht als een vloeistof zonder gestandaardiseerde concentratie.

GHB is typically sold as a clear, odorless liquid in unlabeled bottles with no concentration information. A teaspoon of one batch may contain 1g. A teaspoon of another may contain 4g. The difference between a recreational dose (1–2g) and a dangerous dose (3–4g) and a potentially lethal dose (5g+) may be invisible in the same volume of liquid.

This measurement problem is the primary driver of accidental GHB overdose in recreational users. Tolerance, batch concentration variation, and the combination with alcohol create a lethal uncertainty that no other common substance matches in its unpredictability.

- If you are using GHB: use a precise oral syringe (1ml markings) for measurement. Never drink directly from the bottle. Never measure with spoons.
- If you are helping someone who uses GHB: know the signs of overdose above. Know that 000 is the only emergency number to call for unconsciousness. Know that there is no antidote.
- GHB test kits: available through harm reduction organisations – can verify the presence of GHB but not concentration. Use them.

# Section 1 — What GHB Did to Your Brain

**THE CENTURION — The molecule your brain already makes — used on a scale your brain cannot manage.**

GHB (gamma-hydroxybutyrate) is endogenous. Your brain produces it in small amounts as both a neurotransmitter and a metabolite of GABA. This biological familiarity is part of why GHB's effects feel qualitatively different from other substances — the brain recognizes this molecule. It has receptors specifically designed for it.

Exogenous GHB at recreational and addictive doses operates through two primary mechanisms:

## The two receptor systems that GHB disrupts are:

## * The GABA-B receptor system.
## * The cannabinoid receptor system.

| Receptor system. | At high doses, GHB can cause:<br><br>* Loss of consciousness<br>* Seizures<br>* Respiratory depression (slowed or stopped breathing)<br>* Coma<br>* Death<br><br>If someone who has taken GHB becomes unresponsive or stops breathing, it is a medical emergency. Call for medical help immediately. Naloxone may be used to reverse the effects of an opioid overdose, but it will not work if the person has overdosed on GHB alone. | What this means in withdrawal and recovery. |
|---|---|---|
| GABA-B | Potent GABA-B agonist at | Chronic GABA-B overstimulation causes |

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|---|---|---|
| receptors (primary dependence mechanism). | recreational doses (1–4g). Activates the same receptors as baclofen. Produces profound inhibition of excitatory neurotransmission — sedation, anxiolysis, muscle relaxation, euphoria. | receptor downregulation and reduced endogenous GABA production. When GHB is removed, the brain's primary braking system is severely depleted while glutamate surges unchecked. The result is the most severe acute withdrawal syndrome of any common substance — more abrupt and more dangerous than alcohol withdrawal because of GHB's 3–4 hour half-life and the completeness of GABA-B occupancy. |
| GHB-specifika receptorer (det endogena systemet). | Low doses of GHB (below recreational levels) activate GHB-specific receptors, which are distinct from GABA-B. These receptors modulate dopamine release in mesolimbic circuits — particularly relevant to GHB's mood-elevating and pro-social effects at lower doses. | GHB receptor downregulation in heavy users contributes to the anhedonia, social disconnection, and emotional blunting of GHB recovery. Unlike GABA-B recovery (which is medically urgent and relatively fast once stable), GHB receptor recovery is slower and subtler — contributing to PAWS months after physical withdrawal is complete. |
| Sistema dopaminérgico (secundario). | GHB modulerar indirekt dopamin via både GABA-B- och GHB-receptorvägar – initialt ökar det mesolimbiskt dopamin vid låga doser, men minskar det vid långvarig, hög användning. | Deficit de dopamina durante la recuperación del GHB produce la misma anhedonia y el mismo deterioro de la motivación que se observan en la recuperación de estimulantes y opioides, pero a menudo es eclipsado por el desequilibrio más dramático entre GABA y glutamato durante la abstinencia aguda. La reconstrucción de la dopamina se convierte en el objetivo central a largo plazo una vez que la fase aguda ha pasado. |

## The Half-Life Problem — Why GHB Dependence Is Uniquely Dangerous.

### THE CENTURION — The brain recalibrates to whatever schedule you give it.

GHB has a half-life of approximately 30–60 minutes for the active compound (plasma levels), with a duration of effect of 1.5–3 hours depending on dose. This is dramatically shorter than alcohol (hours), benzodiazepines (hours to days), or opioids (hours to days).

The consequence: a person who uses GHB every 2–3 hours has trained their brain to expect a GABA-B agonist signal at 2–3 hour intervals. The GABA-B receptors have downregulated around this schedule. When a dose is missed — or when the person is asleep and cannot dose — the brain experiences acute withdrawal within 2–4 hours of the last dose.

This creates the defining pattern of severe GHB dependence: the person must wake multiple times per night to dose. They cannot travel on planes. They cannot attend medical appointments, work shifts, or social events that would put them more than 2–3 hours from their supply. Their entire life has been organized around the dosing schedule. And the physical dependence has been building the entire time.

Dit is geen zwakte. Dit is wat de biologie van de GABA-B-receptor doet wanneer deze chronisch wordt overstimuleerd volgens een kort cyclus schema. De valkuil is farmacologisch, niet karakterologisch.

## The GHB Class — Forms and Precursors

### THE CENTURION — GHB, whatever it's called, has the same biological effects.

| Form | What it is. | Vitesse de conversion / délai d'action. | Recovery-specific notes. |
|---|---|---|---|
| GHB (gamma-hydroxybutyrate) | The active molecule — sodium or potassium salt in solution. | Immediate — 15–30 minute onset. | GHB purchased from the street does not have a standardised concentration. Accurate measurement is crucial but is often unavailable. It's a clear liquid with a salty taste, and it's very difficult to measure the dosage accurately. |
| GBL (gamma-butirolacton). | Industrial solvent and direct GHB precursor — converted to GHB in the body within minutes of ingestion. | Faster than GHB — 5–15 min onset; more potent mg-for-mg. | MORE DANGEROUS THAN GHB in several respects: faster onset means dosing errors happen more rapidly; higher potency means smaller margin between dose and overdose; industrial sources have variable purity. The same dependence biology as GHB with amplified risk profile. |
| 1,4-BD (1,4-butanediol) | Industrial solvent converted to GHB via alcohol dehydrogenase in the liver. | Slower than GBL — 20–40 min onset; longer duration. | Because 1,4-BD is converted via ADH (the same enzyme that metabolizes alcohol), ALDH2 variants affect its metabolism and toxicity. Combining 1,4-BD with alcohol is particularly dangerous because both compete for ADH conversion — producing unpredictable plasma levels and delayed overdose. |
| Sodium oxybate (Xyrem – prescription). | Pharmaceutical-grade GHB prescribed for narcolepsy and now some other | Predictable onset — controlled formulation, known | La dipendenza da sodio oxibato è spesso sottovalutata perché i pazienti presumono che i farmaci prescritti siano "sicuri" in termini di dipendenza. La farmacologia è |

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| | conditions. | concentration. | identica a quella del GHB di strada. Se si interrompe Xyrem, discutere la possibilità di una riduzione graduale con il neurologo che ha prescritto il farmaco; l'interruzione improvvisa comporta gli stessi rischi di astinenza del GHB di strada. |

## Who uses GHB and why — The contexts that shape recovery.

### THE CENTURION — The context determines what was being managed and what recovery must address.

GHB use disorder appears across genuinely different populations with different underlying drivers. Understanding which context describes your use determines which additional recovery elements beyond the core GABA reconstruction protocol are most important.

| Use context | GHB was providing. | Additional recovery considerations:<br><br>* [ ] Assess for co-occurring mental health conditions and provide appropriate treatment.<br>* [ ] Address any legal or financial issues that may be impacting recovery.<br>* [ ] Develop a relapse prevention plan.<br>* [ ] Connect with peer support groups or other community resources.<br>* [ ] Consider vocational rehabilitation or educational opportunities.<br>* [ ] Explore creative outlets and hobbies to promote well-being.<br>* [ ] Practice self-care activities such as exercise, healthy eating, and mindfulness.<br>* [ ] Seek professional guidance from a therapist or counsellor if needed.<br>* [ ] Celebrate milestones and acknowledge progress made in recovery.<br>* [ ] Remember that |
|---|---|---|

| | | |
|---|---|---|
| | | **recovery is an ongoing process and setbacks may occur.** |
| Chemsex / nightlife (GHB + crystal meth + mephedrone) | Sexual disinhibition, social belonging, euphoria, extended endurance. GHB's GABA-B activation in this context counterbalances the stimulant anxiety from crystal meth — the two substances are used in combination intentionally. | The chemsex pattern in the compendium (Volume VII, specialist protocols) addresses the sexual-social-chemical complex that GHB often forms part of. Recovery must address the sexual and social context alongside the substance. HIV and STI testing, PrEP status, and sexual health are clinical priorities. |
| Anxiety / self-medication for sleep | GHB produces rapid, profound anxiolysis and sleep initiation — faster and more complete than alcohol for many users. The Sage constitution (slow COMT, chronically elevated stress chemicals) may find GHB extraordinarily effective at quieting a nervous system that will not quiet. | Same as alcohol and benzodiazepine anxiety-use profiles: the anxiety was there before the GHB and will be there after. Anxiety management is the central recovery target. GABA-B sensitization through natural means replaces the pharmacological solution. |
| Athletic / bodybuilding use. | GHB stimuleert de afgifte van groeihormoon bij lage doseringen - wordt door sommige sporters gebruikt als prestatieverbeterend middel en herstelhulpmiddel. Afhankelijkheid ontwikkelt zich geleidelijk naarmate de doses toenemen en de frequentie noodzakelijk wordt om het slapen en herstel te behouden. | La riparazione dell'asse dell'ormone della crescita fa parte del quadro a lungo termine. Le strategie naturali che stimolano l'GH (sonno profondo, digiuno, esercizi HIIT) sostituiscono l'approccio farmacologico. L'ipamorelin e altri GH-secretagoghi sono stati utilizzati come "ponte" sotto la guida di un medico. |
| Narcolepsy management (sodium oxybate). | Sodium oxybate är den standardmässiga behandlingen för narkolepsi med kataplexi. Beroendet som utvecklas är en nödvändig följd av behandlingen, inte ett moraliskt misslyckande. | Discontinuing sodium oxybate requires neurologist involvement because the narcolepsy symptoms—which were being managed—will return upon cessation. This is a clinical decision requiring specialist support, not a self-managed taper. Do not stop without your neurologist. |
| Alcohol harm reduction. | Some people have used GHB explicitly to reduce alcohol consumption — trading one GABAergic substance for another with a different risk profile. The trade-off is complex: | Si GHB se utiliza como sustituto del alcohol, es posible que ambas sustancias causen dependencia al mismo tiempo. Es esencial una evaluación médica de la dependencia |

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|---|---|---|
| | GHB may have a lower organ toxicity profile than alcohol at moderate use, but its dependence liability and overdose risk profile are severe. | combinada de GABA antes de intentar cualquier reducción. |

# Section 2 — Your Genomic Blueprint

GHB recovery has a specific genomic profile that maps directly onto the compendium's GABA system genetics. The same variants that determine alcohol and benzodiazepine risk also determine GHB risk—with one additional consideration unique to GHB's role in chemsex and dopaminergic contexts.

## The DNA Testing Panel for GHB Recovery.

| Gene | What it controls. | Key variant. | GHB recovery relevance. |
|---|---|---|---|
| GABRA2 | GABA-A receptor alpha-2 subunit — baseline inhibitory tone. | rs279858 / rs279845 high-risk haplotype. | THE most important gene for GHB vulnerability. The compendium explicitly identifies GABRA2 high-risk carriers as the 'Hard Fail' profile for combining alcohol with GABAergic agents including GHB. High-risk variants structurally reduce baseline GABAergic inhibitory tone — the brain's brake system runs at chronically low power. GHB powerfully activates this underpowered system. Recovery for this profile: GABA restoration is slower, PAWS is more severe, and natural GABA-building practices are non-negotiable rather than supportive. |
| COMT | Dopamin og stresskjemikalier fjerning. | Val158Met (rs4680) | Sage constitution (Met/Met, slow COMT) is overrepresented in anxiety-driven GHB use — the drug's GABA-B anxiolysis provides relief that a chronically overstimulated nervous system cannot generate. Warrior constitution (Val/Val) appears in chemsex and stimulant-adjacent use — GHB as the counterbalance to stimulant anxiety. Constitution determines recovery strategy direction: Sage needs calm-building; Warrior needs stimulation-management. |
| MTHFR | Metilazione — sintesi dei neurotrasmettitori. | C677T (rs1801133), A1298C (rs1801131) | MTHFR variants create baseline neurochemical deficits that GHB was partially addressing. 5-MTHF supplementation is foundational and |

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|---|---|---|---|
| | | | often produces early visible improvement. GHB also depletes B-vitamins through metabolic pathways — addressing methylation deficits is urgent in early recovery. |
| GABBR1 / GABBR2 | GABA-B receptor subunit genes – the primary receptor that GHB acts on. | Various variants affecting receptor sensitivity and density. | Emerging pharmacogenomics — variants in the GABA-B receptor genes themselves affect individual response to GHB, sensitivity to its effects, and the severity of GABA-B receptor downregulation with chronic use. Limited clinical testing availability currently, but a field worth tracking. |
| DRD2 / ANKK1 | Densidade dos receptores de dopamina D2 — Síndrome da Deficiência de Recompensa. | Taq1A (rs1800497) | A1 allele (reduced D2 density) is relevant to GHB use in the chemsex and nightlife context — the dopaminergic reward from GHB partially filling the RDS gap. Recovery must build dopamine reward architecture or the pull toward any dopaminergic substance (including resuming GHB) will remain biologically driven. |
| SLC6A4 / 5-HTTLPR | Serotonin transporter expression. | Short/short allele. | High anxiety sensitivity in short/short carriers predisposes to GABAergic substance use for anxiety management. GHB recovery must address the serotonin system alongside GABA — 5-HTP and 5-MTHF supplementation support both, as does the contemplative practice architecture in Section 6. |
| CYP2C19 / CYP3A4 | Metabolismo de baclofeno y benzodiazepinas utilizadas en la desintoxicación de GHB. | Poor to ultra-rapid metabolizer variants. | Baclofen is the primary medical intervention for GHB withdrawal management. CYP2C19 and CYP3A4 variants affect baclofen metabolism — poor metabolizers accumulate drug at standard doses; ultra-rapid metabolizers may need higher doses for adequate effect. Inform your detox physician of any known CYP variant status. |
| ADH1B / ALDH2 | Alcohol and 1,4-BD metabolism. | rs1229984 (ADH1B), rs671 (ALDH2) | Specifically relevant for 1,4-BD users — the ADH/ALDH pathway converts 1,4-BD to GHB in the liver. ALDH2*2 carriers who used 1,4-BD alongside alcohol face compounding conversion pathway conflicts and accumulated acetaldehyde toxicity. Liver function testing is particularly important for |

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|---|---|---|---|
| | | | this subgroup. |

# Section 3 — The Detox Protocol

**⚠ Detox from GHB is a medical emergency for anyone taking it more than 3-4 times daily. The risk of seizures and psychosis in the first 12-72 hours is real, unpredictable, and can be fatal without medical management.**

## 3.1 — Why GHB withdrawal is uniquely dangerous.

**THE CENTURION — The Shortest Half-Life, the Fastest Dependence, the Most Abrupt Withdrawal.**

GHB withdrawal is medically distinct from alcohol and benzodiazepine withdrawal in several ways that make it particularly treacherous:

- Speed of onset: Because GHB has a 30–60 minute plasma half-life, withdrawal begins within 2–6 hours of the last dose for dependent users — far faster than alcohol (6–24 hours) or benzodiazepines (12–48 hours). The window from last dose to seizure risk is extremely narrow.
- Unpredictability: The clinical severity of GHB withdrawal does not always correlate predictably with the reported use pattern. People who underreport their use (or whose street GHB had variable concentration) may present in more severe withdrawal than anticipated.
- Psicose: A abstinência de GHB pode causar uma síndrome específica de delírio e psicose que se desenvolve rapidamente e é diferente da síndrome alcoólica de abstinência, caracterizada por agitação, confusão, alucinações visuais e extrema instabilidade autonômica. Isso não é apenas ansiedade. É uma emergência neurológica.
- Treatment complexity: Unlike alcohol withdrawal (benzodiazepines) or opioid withdrawal (buprenorphine), there is no FDA-approved specific treatment for GHB withdrawal. Baclofen (a GABA-B agonist) is the most evidence-supported option and represents the most direct pharmacological match to GHB's primary mechanism. Benzodiazepines are used but are less pharmacologically specific and may require very high doses.

## 3.2 — Opzioni di disintossicazione medica

**THE CENTURION — The Treatment Landscape for the Most Underserved Withdrawal in Medicine.**

| Approach | Mechanism. | Clinical notes. |
|---|---|---|
| Baclofen tapering (preferred where available). | GABA-B agonist — directly substitutes for GHB at the receptor | The most pharmacologically rational approach: baclofen addresses the specific receptor system that GHB has |

| | | |
|---|---|---|
| | level. Longer half-life allows a gradual tapering process with stable blood levels. | disrupted. Doses required are often very high (up to 200–300mg/day) and must be titrated carefully. Not all emergency rooms or detox facilities are familiar with baclofen for GHB withdrawal — you may need to advocate for this specifically. A physician experienced in GHB detox is strongly preferred. |
| Benzodiazepine protocol (most commonly used) | GABA-A agonist — affects the GABA system generally, but targets a different receptor subtype compared to GHB's main target. | High doses of long-acting benzodiazepines (diazepam, chlordiazepoxide) are commonly used because they are available and familiar to emergency physicians. Less pharmacologically specific than baclofen — may require very high doses to control severe GHB withdrawal. Risk of transitioning benzodiazepine dependence. Requires close medical monitoring. |
| GHB withdrawal using pharmaceutical-grade sodium oxybate. | Substitutes the same molecule in a controlled, measured form, then tapers gradually. | The most theoretically elegant approach — same molecule, controlled concentration, known pharmacokinetics. Used in some specialist addiction units. Limited availability outside of countries where sodium oxybate is accessible for this indication. Requires a specialist addiction medicine physician. |
| Fenobarbital. | Barbitúrico de acción prolongada con actividad agonista del receptor GABA-A; su vida media muy larga proporciona niveles estables en el plasma. | Used in specialist settings for complex GABAergic polydrug withdrawal. The extreme half-life (53–118 hours) provides inherent tapering. Requires careful monitoring for respiratory depression. Not first-line but valuable in refractory cases. |
| Supportive medications (adjunctive to above) | Clonidina (per la gestione delle tempeste autonome), antipsicotici per la psicosi, anticonvulsivanti per la prevenzione delle crisi epilettiche, idratazione, gestione degli elettroliti. | GHB withdrawal produces extreme autonomic instability (tachycardia, hypertension, sweating) alongside psychiatric symptoms. Clonidine addresses the autonomic component. Antipsychotics (haloperidol, quetiapine) for delirium and hallucinations. IV fluids and electrolyte monitoring for the profuse sweating and autonomic dysfunction. |

## 3.3 — The Withdrawal Timeline

| Timeline. | Biology. | Symptoms | Medical response. |
|---|---|---|---|
| Hours 2–6 (onset – the medical emergency window). | GHB clears rapidly; GABA-B receptors without ligand; glutamate surging; HPA axis activating. | Anxiety, tremor, sweating, rapid heartbeat, nausea — the onset is fast and can be mistaken for 'normal' anxiety until it escalates. | THIS IS THE MEDICAL INTERVENTION WINDOW. If the person is in a monitored medical setting, this is when baclofen or benzodiazepines should be initiated. If the person is not in medical care and symptoms are appearing: go to emergency department now. |
| Hours 6–24 (peak danger) | Maximum GABA-B deficit; glutamate excitotoxicity; HPA axis in full overdrive; dopamine system dysregulated. | Grave ansiedad, agitación, confusión, alucinaciones visuales y auditivas, delirio, convulsiones, taquicardia, hipertensión, hipertermia: cualquier combinación de estos. | Inpatient medical management required. Baclofen titration or high-dose benzodiazepine protocol. Continuous monitoring. IV access. Seizure precautions. |
| Hours 24–72 (continued medical risk) | Symptoms of withdrawal can sometimes last or get worse before getting better. The GABA-B system may be significantly depleted. Psychiatric symptoms might be noticeable. | Confusione mentale può persistere o raggiungere il picco in questo periodo; agitazione; instabilità autonoma continua; alcuni miglioramenti nei sintomi fisici, ma i sintomi psichiatrici possono essere al massimo. | Continued inpatient monitoring. Antipsychotics for delirium as needed. Gradual medication taper begins when clinically stable. |
| Days 3–7 (stabilization) | Acute withdrawal resolving with medical management; GABA-B receptors beginning to recover; glutamate calming; but significant neurochemical instability remains. | Physical symptoms improving substantially; cognitive confusion clearing; emotional rawness; depression emerging; profound fatigue; insomnia. | Begin supplement protocol (Section 4). Nutritional assessment and replenishment. Transition from acute medical management to a recovery framework. Sleep support becomes a priority. |
| Weeks 1–4 (post-acute) | GABA-B receptor upregulation ongoing; glutamate normalizing; dopamine system beginning to | Anxiety persistente (frequentemente grave nas primeiras 1-2 semanas), depressão, insônia, confusão mental, | Full Recovery Kernel supplement and behavioral protocol. Exercise as soon as physically capable. The anxiety of this phase is the primary relapse driver — |

| | | | |
|---|---|---|---|
| | recover; HPA axis slowly normalizing. | desejos, instabilidade emocional, hipersensibilidade à estimulação. | GABA-supportive interventions are highest priority. |
| Months 1–6 | Significant GABA-B recovery; dopamine and serotonin systems rebuilding; HPA axis normalization; brain adapting to the endogenous GABA system being back in charge. | Gradual improvement with occasional setbacks; withdrawal symptoms are likely; a genuine return to feeling relaxed and enjoying life is within reach. | The natural GABA architecture from Section 6 becomes increasingly effective as the endogenous system recovers. PAWS episodes require the full interrupt protocol and medical contact if severe. |
| Months 6–18+. | Densité des récepteurs GABA-B largement rétablie ; système dopaminergique normalisé ; axe HPA stable ; système de récepteurs GHB en voie de récupération. | Near-normal anxiety tolerance and stress resilience; natural pleasure and calm are genuinely accessible; episodic cravings are primarily context-dependent. | La arquitectura de "adicción natural" del compendio (C-28) se vuelve completamente operativa. El objetivo a largo plazo es construir una vida basada en un sistema GABA recuperado, no en la sustancia que lo estaba gestionando temporalmente. |

## 3.4 — Post-Acute Withdrawal Syndrome in GHB Recovery.

**⚠ GHB withdrawal symptoms can persist for 12–18 months and may be among the most prolonged withdrawal syndromes in addiction medicine. The anxiety is real. It is neurological. It will resolve.**

PAWS in GHB recovery is characterized by episodic severe anxiety, sleep disruption, cognitive fog, cravings that appear suddenly and intensely, and emotional volatility that can appear weeks or months into recovery without warning. The GABRA2 high-risk genotype is associated with more severe and prolonged PAWS.

GHB PAWS is particularly challenging because the anxiety component is severe — often more severe than PAWS from other substances — and because the drug that would relieve it is the one the person is in recovery from. Managing this anxiety through non-pharmacological and supplement means requires the most robust implementation of the GABA restoration protocols in this guide.

PAWS interrupt: return to Protocol 01 (Shelter). Execute 4-7-8 breathing immediately and repeatedly. Contact your medical support. Increase magnesium glycinate dose. Use L-theanine. If psychotic symptoms appear (hallucinations, severe confusion) at any point in the recovery period — this is a medical emergency, not a PAWS episode. Go to emergency services.

# Section 4 — Biological Reconstruction

GHB recovery requires the reconstruction of three systems: the GABA-B receptor system (the primary target), the dopamine system (secondary), and the HPA axis (stress response normalization). The methylation foundation underlies all three. This is architecturally similar to alcohol recovery but with specific additions for the GABA-B receptor subtype and the unique neurochemistry of GHB's own receptor system.

Diskussieren Sie dies mit Ihrem Arzt, bevor Sie beginnen, insbesondere angesichts des potenziellen Risikos von Wechselwirkungen zwischen Nahrungsergänzungsmitteln und Medikamenten während einer Medikationseinstellung. Einige Nahrungsergänzungsmittel (Zitronenmelisse, Baldrian, hochdosiertes Taurin) können die Sedierung verstärken, wenn sie in Kombination mit Baclofen oder Benzodiazepinen verwendet werden.

## 4.1 — The Methylation Foundation

GHB, GBL, and 1,4-BD all place metabolic demands on the methylation system. The production and breakdown of GABA itself is B6-dependent. Addressing methylation deficits is the non-negotiable first layer.

| Supplement | Dose. | Timing. | GHB-specifika noteringar. |
|---|---|---|---|
| 5-MTHF (L-methylfolate) | 400–1000 mcg daily. | Breakfast. | GABA synthesis is folate-dependent. 5-MTHF provides activated folate for the full neurotransmitter synthesis pathway. Start at 400mcg and increase slowly — rapid methylation initiation can temporarily worsen anxiety in some, particularly relevant in early GHB recovery when anxiety is already severe. |
| Methylcobalamin (B12) | 1000–2000mcg | Morning, sublingual. | Works synergistically with 5-MTHF. GHB/GBL/1,4-BD metabolic burden depletes B12. Sublingual absorption bypasses GI conversion. Essential for nervous system repair and myelin integrity. |
| P5P (active B6) | 25–50mg | Morning. | THE most critical cofactor for GABA synthesis specifically. Glutamate decarboxylase — the enzyme that converts glutamate |

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| | | | to GABA — is B6-dependent. In GHB recovery, where the GABA system needs to rebuild its own production capacity, P5P is the enabling cofactor. Use P5P form only — standard pyridoxine does not convert adequately in some individuals. |
| Magnesium glycinate. | 400–600mg elemental | Morning and evening. | Magnesium stabilises GABA-A receptors and is required for GABA synthesis, and it reduces NMDA glutamate excitotoxicity. In GHB recovery, where the GABA-glutamate imbalance is the central pathological mechanism, magnesium addresses the imbalance from multiple angles simultaneously. Highest-priority single supplement in GHB recovery. |
| Zinc. | 20–30mg daily. | Evening with food. | Zinc is involved in GABA synthesis and receptor function. GHB/GBL metabolism depletes zinc. Also supports immune function and mood regulation. Take with food to prevent nausea; do not take with calcium supplementation (competitive absorption). |

## 4.2 — GABA-B System Restoration

### THE CENTURION — Supporting the system that was borrowed against, specifically.

Up-regulation of GABA-B receptors is a structural process that occurs through abstinence, over time, and is supported by neuroplasticity involving BDNF and appropriate cofactors. Supplements support the conditions for this up-regulation; they do not directly replace GHB's GABA-B activation. The recovery is biological and time-dependent.

**⚠ CAUTION WITH SUPPLEMENT TIMING: If you are currently on a baclofen or benzodiazepine taper, discuss all supplements with your physician before adding them. GABA-supporting supplements can enhance sedation and respiratory depression when combined with these medications. Add supplements only once the taper is complete or under physician guidance.**

| Supplement. | Dose. | Timing. | The role of a psychiatrist in |
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| | | | **GHB recovery can include:**<br><br>*** Assessing the individual's overall mental and physical health.**<br>*** Developing a comprehensive treatment plan that addresses both the addiction and any co-occurring mental health conditions.**<br>*** Providing medication management, including prescribing medications to manage withdrawal symptoms, cravings, and other psychiatric disorders.**<br>*** Offering psychotherapy or counselling to help the individual understand their addiction, develop coping skills, and address underlying issues that may have contributed to their substance use.**<br>*** Monitoring the individual's progress and making adjustments to the treatment plan as needed.**<br>*** Providing support and education to the individual and their family members.**<br><br>**A psychiatrist can also work with other healthcare professionals, such as addiction specialists, therapists, and social workers, to provide a coordinated approach to care.** |
| Magnesium glycinate (repeated — highest priority) | 300–400mg elemental | Morning and evening. | Repeated from Section 4.1 because it is the single most important supplement in GHB recovery. NMDA antagonism, GABA receptor stabilization, GABA synthesis support, anxiety reduction. Start on Day 1 of the post-detox recovery phase. |
| Taurine | 1–3g daily. | With meals. | Taurine activates GABA-A receptors and has neuroprotective effects through NMDA modulation. Acts as a |

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| | | | GABA-mimetic amino acid. Particularly relevant in GHB recovery for its dual role: calming the glutamate surge AND providing hepatoprotection — GBL and 1,4-BD are processed through the liver and taurine supports hepatic function. |
| L-Theanine | 200–400mg | Efter behov vid ångest, eller 2-3 gånger dagligen under akut fas. | GABA and glycine modulation; promotes alpha-wave brain states; non-sedating anxiolytic without dependence. This is particularly valuable in GHB recovery because the anxiety is the primary relapse driver and must be managed without using benzodiazepines (which carry their own dependence risk) or returning to GHB. L-theanine provides a genuine anxiolytic effect without addiction liability. |
| Inositol. | 4–18g daily (increase the dose gradually). | Divided with meals. | Modulates GABA and serotonin receptor sensitivity; reduces anxiety; anti-obsessional properties relevant to the obsessive-compulsive quality of GHB cravings in early recovery. High dose required for psychiatric effect — start at 4g and increase by 2g weekly. GI tolerance generally good but monitor. |
| Lemon balm (Melissa officinalis) | 300–600mg standardized extract. | Evening – avoid if taking sedating medications. | Inhibidor da GABA transaminase: retarda a degradação de GABA, aumentando o tônus disponível de GABA. Tem um efeito ansiolítico leve e há evidências de sua eficácia em transtornos de ansiedade. É mais adequado após a conclusão da redução da dose do medicamento; não deve ser combinado com baclofeno ou benzodiazepínicos sem orientação médica. |
| Fosfatidilserina. | 100–300mg | Evening. | Reduces evening cortisol — the HPA axis rebound in GHB recovery produces elevated cortisol that prevents sleep onset and amplifies anxiety. Phosphatidylserine directly addresses the HPA axis component. Also supports |

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| | | | neuronal membrane health relevant to receptor recovery. |
| NAC (N-Acetylcystein). | 600–1800mg daily in divided doses. | Morning and evening with food. | Restores glutamate homeostasis in the nucleus accumbens. Particularly relevant in GHB recovery because GHB's mechanism involves glutamate suppression — the glutamate surge in withdrawal represents the system rebounding from chronic suppression. NAC helps normalize the glutamate system. Also provides liver support through glutathione precursor function — relevant for GBL and 1,4-BD users whose liver processed the conversion. |

## 4.3 — Dopamine and Serotonin Reconstruction

Once the acute GABA/glutamate crisis has passed, the dopamine and serotonin systems that were secondarily disrupted by GHB become the primary targets for the long-term recovery phase.

| Supplement | Dose. | Timing. | Notes |
|---|---|---|---|
| 5-HTP | 50–150mg | Evening. | Serotonin precursor — supports mood and sleep. GHB use suppresses serotonin tone over time. 5-HTP in the evening supports serotonin synthesis and conversion to melatonin, addressing both mood and sleep simultaneously. Start at 50mg. Do not combine with SSRIs or SNRIs without physician guidance. |
| L-Tyrosine / NALT | 500–1500mg | Good morning, no protein today. | Precursor de la dopamina: ayuda a mejorar el estado de ánimo y la motivación. La anhedonia y el déficit de motivación en la recuperación del GHB (especialmente en contextos de "chemsex" donde el GHB formaba parte de una experiencia dopaminérgica) pueden mejorarse con suplementos precursores de la dopamina. Comience después del día 7, una vez que esté |

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| | | | comiendo regularmente. |
| Rhodiola rosea | 300–600mg | Morning. | Adaptogen — modulates dopamine and serotonin; reduces fatigue; regulates cortisol levels. Particularly useful during weeks 2-8, when fatigue and lack of motivation are common, but a precursor stack alone is not enough. Do not take late in the day — it's stimulating. |
| Omega-3 (EPA + DHA) | 2–4g daily | With food, in the morning. | Supports BDNF expression, reduces neuroinflammation, provides membrane structural support for receptor recovery. EPA-dominant formulation for anti-inflammatory and mood effects. Essential in the first 6 months alongside all other recovery work. |

## 4.4 — Sleep Reconstruction

**THE CENTURION — The system that controlled your sleep schedule must now rebuild itself.**

For people who are dependent on GHB and who were taking it at night to avoid withdrawal symptoms while they slept—which is a key sign of serious GHB addiction—restoring healthy sleep patterns is the most important and also the most difficult part of getting better.

The brain that was using GHB as a sleep-induction tool has not practiced the natural sleep initiation process for months or years. The endogenous GABA-B activity that normally facilitates sleep onset has been chronically suppressed. The sleep architecture damage is profound: no natural deep sleep, no REM, no circadian entrainment — just GHB-induced unconsciousness on a 2–3 hour cycle.

- Melatonin: 0.5–1mg sublingual, 30–60 minutes before your desired bedtime. A low dose is crucial – high doses can disrupt your circadian rhythm rather than supporting it. Add tart cherry extract (480mg) for additional melatonin precursor support.
- Magnesium glycinate: 400mg elemental 1 uur voor het slapengaan - het belangrijkste slaapsupplement bij herstel van GHB, vanwege de stabiliserende werking op de GABA-receptoren en de vermindering van glutamaat.
- Glycine: 3–5g, 30 minutes before bed — reduces core body temperature, the primary physiological sleep trigger. NMDA co-agonist that supports the GABA-glutamate rebalancing needed for sleep architecture.

- L-Theanine: 200–400mg evening — promotes alpha waves without causing drowsiness. Reduces the anxious hyperarousal that prevents sleep onset in early GHB recovery.
- Fosfatidilserina: 100–200 mg al ser, reduceix el cortisol nocturn que impede l'inici del son.
- Inositol: 4–8g in the evening — modulates serotonin and GABA receptors; supports the transition to sleep without causing sedation.

Protocol 17 (Sleep Architecture)

[This protocol is designed to help restore healthy sleep patterns after periods of disrupted sleep, such as those experienced during substance use or withdrawal.]

* **Understanding Sleep Architecture:**
  * Normal sleep consists of cycles lasting approximately 90-120 minutes.
  * Each cycle includes stages of light sleep, deep sleep (slow-wave sleep), and REM sleep.
  * Disruptions to these cycles can lead to fragmented sleep, daytime fatigue, and other health problems.

* **Addressing GHB-Specific Considerations:**
  * Expect waking during the night for weeks or months. The brain was trained to wake every 2–3 hours. Do not interpret this as failure.
  * Do not dose GHB when you wake.
  * Use the 4-7-8 breath and return to sleep.
  * The pattern will gradually extinguish with consistent practice.

* **General Strategies for Restoring Sleep Architecture:**
  1. **Establish a Regular Sleep Schedule:** Go to bed and wake up at the same time each day, even on weekends, to regulate your body's natural sleep-wake cycle (circadian rhythm).
  2. **Create a Relaxing Bedtime Routine:** Engage in calming activities before bed, such as taking a warm bath, reading a book, or listening to soothing music. Avoid screen time (phones, tablets, computers) for at least an hour before bed, as the blue light emitted from these devices can interfere with sleep.
  3. **Optimize Your Sleep Environment:** Ensure your bedroom is dark, quiet, and cool. Use blackout curtains, earplugs, or a white noise machine if necessary. A comfortable mattress and pillows are also essential for good sleep.
  4. **Limit Caffeine and Alcohol Intake:** Avoid caffeine and alcohol consumption in the hours leading up to bedtime, as they can disrupt sleep patterns.
  5. **Regular Exercise:** Engage in regular physical activity, but avoid intense workouts close to bedtime. Aim for at least 30 minutes of moderate-intensity exercise most days of the week.
  6. **Manage Stress and Anxiety:** Practice relaxation techniques such as meditation, deep breathing exercises, or yoga to reduce stress and anxiety levels, which can interfere with sleep.
  7. **Consider Cognitive Behavioral Therapy for Insomnia (CBT-I):** CBT-I is a structured program that helps individuals identify and change thoughts and behaviors that contribute to insomnia. It is considered the gold standard treatment for chronic insomnia.

8. **Medication:** In some cases, medication may be prescribed to help improve sleep. However, it is important to discuss the potential risks and benefits of medication with a healthcare professional.

* **Important Considerations:**
    * If you are experiencing persistent sleep problems, consult with a doctor or other qualified healthcare provider.
    * Do not self-treat sleep disorders without medical supervision.
    * Medical help is not failure.

## 4.5 — Peptide Support Layer

The following peptides have mechanistic relevance to GABA system recovery, neuroinflammation, and the specific neurotoxic effects of GHB and its precursors. Physician discussion required.

| Peptide. | The mechanism in GHB recovery involves several factors, including:<br><br>* Metabolism of GHB by enzymes in the liver.<br>* Excretion of GHB and its metabolites in the urine.<br>* Reversal of GHB's effects on the brain by opioid antagonists such as Naloxone.<br>* Supportive care to manage withdrawal symptoms. | Timing. | Notes |
|---|---|---|---|
| BPC-157 | Modulation of the GABAergic system — has demonstrated specific modulation of GABA-B pathways in research; normalisation of dopamine and serotonin systems; repair of the gut-brain axis; neuroprotection from excitotoxic injury. | Day 1 of the post-detox phase — daily. | BPC-157's GABA-B modulating properties make it uniquely relevant in GHB recovery — more so than in any other guide in this series. Animal models show BPC-157 counteracting GABAergic withdrawal symptoms. Oral administration is appropriate. The gut-brain repair is relevant given that GBL and 1,4-BD are GI-absorbed precursors that damage the intestinal lining. |

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| Selank | GABA and serotonin modulation; anxiolytic without dependence; reduces anxiety without sedation; enkephalin modulation. | Weeks 1–12 — daily or as needed for anxiety. | Selank is the most relevant peptide for managing anxiety during GHB recovery because: (1) severe anxiety is the main symptom of Post-Acute Withdrawal Syndrome (PAWS) and the primary cause of relapse; (2) Selank reduces anxiety through a non-addictive mechanism, which is crucial when the substance being abused already affects the GABA system. Intranasal administration. Non-sedating. No risk of dependence. |
| Epitalon | Sleep architecture and pineal pathway support; telomere protection; circadian rhythm normalization. | Weeks 1–6 — 10-day cycle. | The nocturnal dosing pattern of severe GHB dependence devastates circadian rhythm at the neurological level. Epitalon directly supports the pineal pathway that GHB's sleep-induction mechanism was replacing. Critical in the first 6 weeks when sleep architecture is at its most disrupted. |
| Semax | BDNF upregulation; cognitive repair; dopamine modulation; prefrontal cortex support. | Weeks 2 onwards. | The cognitive fog of GHB recovery — particularly the 'G fog' described by heavy users — reflects prefrontal cortex dysfunction from chronic GABA-B overstimulation. Semax's BDNF-driven prefrontal support is relevant. Begin after the most acute phase has passed. Intranasal. |

# Section 5 — The GHB Craving — Understanding and Interrupting.

**THE CENTURION — Les désirs liés au GHB ont une composante physiologique, contrairement à la plupart des autres substances.**

GHB cravings in early recovery have a quality that distinguishes them from most other substances: they can be accompanied by genuine physical anxiety — not just psychological wanting but a body-level alarm signal that mirrors early withdrawal. The GABA-B system's extreme sensitivity in early recovery means that even a PAWS episode can feel physically identical to the beginning of withdrawal. The brain's response is to seek the substance that reliably ended the physical alarm in the past.

Understanding this distinction is critical: the craving-anxiety is NOT evidence that you are in medical danger. It is the recovering GABA system oscillating through periods of lower output as it recalibrates. The tools below address both the psychological and physiological components.

## GHB Craving Types and Their Interrupts

| Tipo di desiderio. | What it feels like. | Interrupción inmediata. |
|---|---|---|
| Craving driven by anxiety (most common in early recovery). | Il sistema nervoso invia ciò che sembra essere un allarme di astinenza: non una voglia psicologica, ma un panico fisico. Battito cardiaco accelerato, sudorazione, la certezza assoluta che sta per accadere qualcosa di terribile se non si assume la dose. | 4-7-8 breathing — immediately and repeatedly. This is not a metaphor for 'try to calm down.' It is a vagus nerve activation protocol that provides genuine GABA-adjacent calming. 400mg L-theanine. 400mg magnesium if not taken today. Cold water on face and wrists. This is a physiological intervention for a physiological alarm. |
| Sleep-context craving (the nocturnal trigger). | Sveglia dopo 2-3 ore dall'inizio del sonno - all'orario previsto dal precedente schema di dosaggio - con ansia acuta, l'aspettativa condizionata di GHB e il sistema di allarme del corpo che si attiva in assenza della dose prevista. | Prepare for this before sleep:<br>* 4-7-8 breathing, ready to go.<br>* Magnesium taken before bed.<br>* Glycine taken.<br><br>When you wake:<br>* Do not check the time if possible.<br>* Execute 4 cycles of 4-7-8 immediately.<br>* Do not get out of bed unless necessary.<br><br>The waking will gradually shift |

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| | | later and eventually disappear as the circadian clock re-entrains. |
| Chemsex / social context craving. | Environmental cues triggers — specific music, people, venues, sexual contexts, or situations associated with GHB use. Can produce sudden intense craving with minimal internal warning. | Environmental exit — leave the context if possible. Call your support contact immediately. The cue-triggered craving requires the cue environment. Change the environment first, interrupt the craving second. Protocol 05 (environmental redesign) should have addressed the highest-risk cue environments — but anticipate that novel cues will arise. |
| Anhedonia, craving. | A flat, colourless experience where the world holds no interest, and GHB is remembered as the only thing that made everything feel electric and vivid. | This is the dopamine deficit speaking, not the GABA deficit. The response is the natural reward architecture (Section 6): vigorous exercise, cold exposure, social connection, creative engagement. The flatness is temporary and resolves as the dopamine system recovers — usually months 2–4. |

### THE GHB CRAVING INTERRUPT — 5-MINUTE PROTOCOL.

1. Anxiety-driven (physiological alarm)?
   Sleep-context (nocturnal waking)?
   Social cue (environmental trigger)?
   Anhedonia (flatness)?
   The type determines the primary response.
2. EXECUTE 4-7-8 BREATHING. Complete at least four cycles. For cravings triggered by anxiety, perform six cycles. This is your main non-pharmaceutical intervention for the GABA system. It's effective and reliable.
3. TAKE YOUR ANXIETY SUPPLEMENTS. L-theanine 400mg. Magnesium glycinate if not already taken. These are not placebos — they are GABA-supporting compounds that address the neurochemical deficit underneath the craving.
4. CHANGE YOUR PHYSICAL ENVIRONMENT. For social/cue cravings: leave the environment before doing anything else. For anxiety cravings: move to a different room, go outside, change your physical position. The environment is part of the circuit.
5. MAKE CONTACT. Text or call your support person, recovery contact, or 13 11 14 line. GHB cravings — especially the anxiety-driven type — lose much of their power in connection. The isolation that GHB's around-the-clock dosing schedule creates is part of the dependence architecture. Connection is its antidote.

6. EXECUTE YOUR NATURAL GABA ACTIVATION. From Section 6: breathwork practice, cold water exposure, gentle movement, music at 60–70 BPM. You are activating the calming system through its natural inputs.
7. Trigger, type, intensity (0-10), action, outcome. After three weeks, this data will reveal your craving pattern with a precision that no therapist session can match.

# Section 6 — Porting the Addiction: Building Your Own GABA.

### THE CENTURION — Centurian Paper C-28 Applied to GHB Recovery — The Internal Off Switch.

GHB gives you something specific. It provides a nervous system that will finally stop. Not partial relief, not reduced anxiety — complete quiet. The GABA-B activation that GHB produces at recreational doses creates a state of neurological stillness that the anxious, overstimulated nervous system experiences as profound relief.

Il framework del Recovery Kernel afferma che lo stato di benessere è non solo accessibile senza GHB, ma è più accessibile, più sostenibile e più autenticamente tuo quando costruito attraverso i sistemi interni del cervello. Ciò che il GHB faceva dall'esterno, il sistema GABA ripristinato può farlo dall'interno. La domanda è quali condizioni lo innescano.

This is the natural addiction architecture for GHB recovery: not focusing on intensity (the stimulant effect), or endorphin warmth (the opioid effect), or anandamide flow (the cannabis effect) — but building the internal capacity for genuine nervous system stillness. Your own GABA. On demand. Without a bottle.

## Natural GABA-B and GABAergic State Activation.

| Metode aktivasi. | GABA effect intensity. | How to implement | GHB recovery specific notes: |
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| | | | * Seek immediate medical attention if experiencing severe withdrawal symptoms such as seizures or hallucinations.<br>* Hydration is crucial during GHB withdrawal, so drink plenty of fluids.<br>* Nutrient deficiencies are common after prolonged GHB use, so consider a multivitamin supplement.<br>* Support groups and therapy can be beneficial for addressing underlying issues that may have contributed to GHB use.<br>* Be aware of the risk of relapse and develop coping strategies to manage |

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| | | | **cravings.**<br>*** Naloxone is not effective in reversing GHB overdose.**<br>*** Recovery Kernel is a resource available to help with GHB addiction recovery.**<br>*** If you or someone you know needs help, visit findahelpline.com.**<br>*** COMT and DRD2 are genes that may be involved in GHB addiction vulnerability.**<br>*** MTHFR is a gene that can affect the metabolism of certain medications used in GHB withdrawal management.**<br>*** A comprehensive assessment by a healthcare professional is essential for developing an individualized recovery plan.**<br>*** Blueprint is a program designed to help individuals overcome substance abuse, including GHB addiction.** |
| Extended exhale breathwork (4-7-8, box breathing, coherent breathing). | High — direct vagus nerve / parasympathetic / GABAergic activation. | Daily practice, minimum 10 minutes in the morning and 10 minutes in the evening. Use as needed for anxiety. 4-7-8 breathing: inhale for 4 seconds, hold for 7 seconds, exhale for 8 seconds. Repeat. | This is the single most important natural GABA-activation tool for GHB recovery. It is not a metaphor — extended exhale breathing measurably increases GABA tone, activates GABA-B receptors via vagus nerve pathways, and reduces cortisol within minutes. The compendium's Protocol 01 breath practice is this. It should be practiced daily until it is as automatic as the former dosing schedule. |
| Exposure to cold water. | High — parasympathetische Aktivierung + Endorphinausschüttung + anschließende anhaltende Ruhe. | Cold shower (30–90 seconds, gradually increasing to full cold). Cold plunge (2–3 minutes below 15°C after acclimatisation). Begin with facial immersion in cold water. | The dive reflex — activated by cold water on the face — is one of the fastest available parasympathetic triggers. It produces immediate heart rate reduction and GABA-mediated calming within seconds. For GHB recovery, where the anxiety spikes are sudden and intense, cold water provides a rapid-response calming tool that has no addiction liability and builds tolerance in the healthy |

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| | | | direction. |
| Deep meditation – specifically absorption states. | High (with practice) — measurably increases GABA and produces GABA-B-adjacent states. | Dharma Recovery (Protocol 04, main Compendium). MBSR.<br>Yoga nidra.<br>Structured meditation with sustained attention on one object for 20+ minutes. | Research on long-term meditators shows significantly elevated GABA tone and GABA-B receptor sensitivity — the internal equivalent of low-dose GHB. This is not accessible in weeks — it builds over months of consistent practice. But the GHB user's former capacity for the 'gone' state (the deep sedation GHB produced) means their nervous system has experienced the territory, and the contemplative path maps to it. Protocol 04 in the compendium is the roadmap. |
| Sustained aerobic exercise. | High — GABA release, BDNF, endorphin, cortisol normalizatio n. | Running, swimming, cycling, rowing — 30–60 minutes at moderate intensity. Daily where possible. | Sustained aerobic exercise increases GABA synthesis and release. It also generates BDNF — the primary driver of GABA-B receptor upregulation through neuroplasticity. Exercise is simultaneously a natural GABA activator AND the primary mechanism for accelerating the receptor recovery that makes natural GABA work better. This is not optional for GHB recovery. |
| Yoga (especially yin and restorative) | Moderate — parasympath etic activation; increased GABA levels have been observed in research. | Yin yoga, restorative yoga, yoga nidra — 45–90 minutes, 3+ times per week. These practices are different from vigorous yoga; the stillness and sustained poses are key. | A study published in the Journal of Alternative and Complementary Medicine showed a 27% increase in GABA levels following a yoga session compared to walking. For GHB recovery specifically, restorative yoga creates the parasympathetic, neurologically quiet state that GHB was providing — through the nervous system's own pathways. The Sage constitution particularly responds to this modality. |
| NSDR — Non-Sleep Deep Rest. | Moderate-High — GABA and dopamine; parasympath etic; measurable neurological rest without | 20–30 minute yoga nidra or NSDR protocol (lying still, guided attention on body). Daily, ideally post-lunch. | NSDR addresses two GHB recovery needs simultaneously:<br>(1) it activates the parasympathetic / GABA calming system;<br>(2) it produces dopamine replenishment comparable to sleep without requiring sleep. |

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| | sleep. | | For someone whose sleep architecture is recovering from months of nocturnal GHB dosing, NSDR provides restoration that normal sleep may not yet deliver. |
| Nature immersion. | Moderate — sustained parasympathetic activity; cortisol reduction; GABA-related calming. | 20+ minutes daily in a natural setting. Forest, ocean, park – any natural environment with reduced artificial stimulation. | Nature environments reduce cortisol levels and activate the parasympathetic nervous system through sensory inputs that have evolved alongside human nervous system development. For a chronically anxious nervous system, like the one that GHB was managing, nature provides the sensory environment in which GABA-mediated calmness is most easily achieved. |

## The Chemsex Context — Additional Recovery Architecture

### THE CENTURION — When GHB Was Part of a Larger Chemical and Sexual Pattern.

For those whose GHB use was embedded in chemsex — the combination of GHB, crystal methamphetamine, and mephedrone in sexual contexts that characterizes a significant portion of GHB use disorder in LGBTQ+ communities — the recovery architecture requires additional elements beyond the core GABA restoration protocol.

- Salute alla salute sessuale: i test per l'HIV e la valutazione dello stato di PrEP sono priorità mediche. Il protocollo specialistico del compendio per la prevenzione dell'HIV/IST nel contesto dell'uso di sostanze (Volume VII, serie di protocolli 35S) si applica direttamente. Il documento "Centurian" sul chemsex nel compendio affronta esplicitamente l'architettura firewall dell'HIV.
- Sexualità durante il recupero: la disinibizione che GHB (e metanfetamine) fornivano in contesti sessuali spesso serviva a gestire vere e proprie ansie, vergogna o difficoltà nell'essere autenticamente vulnerabili. Il recupero richiede lo sviluppo della capacità di vivere esperienze sessuali sobrie; per molti utenti di chemsex, ciò richiede un supporto terapeutico specifico, non solo la cessazione dell'uso di sostanze.
- The social architecture: chemsex communities form tight social bonds organised around substance use. Recovery often requires building a parallel social world—a sober LGBTQ+ community, recovery-oriented spaces—that is not in conflict with the sexual identity that the chemsex community provided belonging for.
- MDMA/mephedrone recovery alongside GHB: In chemsex polysubstance use, mephedrone and MDMA components require separate attention — the

serotonin system damage of these substances is addressed in the MDMA-specific sections of this guide series.

# Section 7 — Navigating the Recovery Kernel: Your Protocol Map.

| Fase. | Protocols | Primary focus | GHB-specifische aantekeningen |
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| Medical Safety (Days 0–7 for dependent users) | Servicio de urgencias / desintoxicación hospitalaria, no es el Compendio. | Prevention of seizures; baclofen or benzodiazepine protocol; management of delirium; monitoring of vital signs. | The Recovery Kernel framework does not begin here for dependent users. Medical management begins here. Once medically stable and through acute withdrawal, return to this guide and start at Biological Foundation. |
| Biological Foundation (Days 1–14 post-detox) | Protocol 07, Protocol 09, Protocol 17, Protocol 18. | Magnesium, B6, methylation stack, sleep architecture, nutrition, gut repair. | Magnesium glycinate on Day 1. P5P on Day 1. 5-MTHF on Day 1. These are not optional foundations — they are the enabling conditions for GABA system recovery. Protocol 18 (nutrition) is particularly important given the nutritional disruption of chronic GHB use and the metabolic demands of GBL/1,4-BD conversion. |
| Genomic Mapping (Weeks 1–4) | Section 15 (Compendium), Protocol 11 | GABRA2 assessment, COMT constitution, MTHFR, CYP2C19. | GABRA2 high-risk variant is the single most important genomic finding for GHB recovery — it explains the severity of dependence, the severity of withdrawal, and the severity of PAWS. Get this test as early as possible. CYP2C19 is relevant if baclofen was used and any ongoing dosing adjustments are being made. |
| GABA Restoration (Weeks 1–12 — the central phase) | Protocol 04, Protocol 01 craving interrupt, Section 4 and 5 of this guide. | Breathwork practice, cold exposure, yoga, magnesium stack, anxiety management, sleep architecture. | Unlike most other substances where the central phase is 4–8 weeks, GHB's GABA-B receptor upregulation is slower and more prolonged. The GABA restoration phase genuinely takes 3–6 months. Consistency in breathwork, exercise, and supplement protocol during this entire window is what determines PAWS severity. |
| Behavioral and Environmental Architecture (Weeks 2– | Protocol 02, Protocol 05, Protocol 08, Protocol 13 | Cue removal, addiction transfer prevention, social architecture redesign, trigger mapping. | The environmental triggers for chemsex (specific venues, social networks, apps) are among the most potent cue-triggered craving environments in addiction medicine. Protocol 05 (environmental |

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| 12) | | | redesign) requires thoroughness regarding both digital environments (apps, chats, groups) and physical locations. |
| Chemsex Recovery (if applicable — Months 1–12) | Protocol 35S series (Volume VII), sexual health protocols. | HIV/STI testing and prevention; sexual health in recovery; sober LGBTQ+ community building. | If GHB was embedded in chemsex, the specialist protocols in Volume VII of the main Compendium address the specific intersections of substance use, sexual health, and community identity that standard addiction protocols do not cover. |
| Identity and Meaning (Months 2–12) | Protocol 20, Protocol 21, Protocol 23, C-28 | Rekonstruksi identitas; diri tanpa sistem saraf yang dikelola secara kimiawi; tujuan. | For long-term dependent GHB users, the substance organized daily life so completely — every 2–3 hours — that removing it creates not just neurochemical withdrawal but temporal and identity disorientation. Protocol 20 (identity reconstruction) addresses who you are when the dosing schedule is no longer the organizing principle of your day. |
| Long Game (Months 6–18+) | Protocol 24, Protocol 25, C-28, C-31 | Purpose architecture; natural GABA system fully operational; the life that the recovered nervous system can inhabit. | La recuperación del receptor GABA-B, que se completa alrededor de los meses 12 a 18, no es solo la resolución de síntomas; es un nuevo punto de partida. El sistema nervioso que antes dependía de GHB cada 2 o 3 horas ahora funciona con mecanismos propios. Esa es una transformación real. Requiere ser algo hacia lo que se trabaja, no simplemente algo por lo que esperar. |

# Section 8 — The Medical Support Layer

La dipendenza da GHB è una delle aree meno servite nella medicina della dipendenza. Molti medici del pronto soccorso, medici di base e persino specialisti della dipendenza hanno poca esperienza nella gestione dell'astinenza da GHB e nelle opzioni farmacologiche più appropriate per essa. Le informazioni seguenti sono pensate per aiutarvi a sostenere un'assistenza adeguata.

## Baclofen — The Most Evidence-Supported GHB Withdrawal Treatment.

Baclofen is a GABA-B agonist — it acts on the same receptor that GHB primarily activates. This makes it pharmacologically the most specific available treatment for GHB withdrawal. Key points to discuss with your physician:

- Doses required for GHB withdrawal management are often much higher than doses used for muscle spasticity (baclofen's approved indication) — up to 200–300mg/day in divided doses. Standard spasticity doses (10–30mg) may be inadequate.
- Titration should be guided by withdrawal symptom severity — a validated tool called the CIWA-Ar (alcohol) can be adapted, but ideally the CGWS (Gamma-Hydroxybutyrate Withdrawal Scale) is used if your facility is familiar with it.
- Reduzierung der Baclofen-Dosis nach Stabilisierung: Sobald die Entzugserscheinungen unter Kontrolle sind, wird die Baclofen-Dosis schrittweise reduziert. Die Reduzierung sollte langsam erfolgen – über Wochen bis Monate, abhängig von der Schwere des Abhängigkeitsproblems.
- Si votre médecin urgentiste ne connaît pas le baclofène pour le sevrage de la GHB : la littérature médicale qui soutient cette approche est disponible ; les articles clés comprennent Addolorato et al. (2009) et Bell et al. (2011). Il est approprié de consulter un spécialiste en médecine de l'addiction.

## Medications for Co-Occurring Conditions.

- Anxiety (post-detox): Buspiron (a non-addictive serotonin 1A partial agonist) is appropriate for persistent anxiety during GHB recovery, and it does not carry the same risk of dependence as benzodiazepines. It takes 2–4 weeks to achieve full effect. CYP3A4 metabolized.
- Depression: MTHFR-variant depression responds to 5-MTHF before prescription intervention is necessary — assess response to 5-MTHF for 4–8 weeks. SSRIs if clinically indicated, with CYP2D6/2C19 considerations for selection.

- Sleep: Quetiapine (low dose, 25–50mg) may help improve sleep patterns in cases of severe insomnia related to GHB use. Use should be limited in time. It's not the preferred initial treatment, but it can be appropriate when other methods are not effective.
- Narcolepsy (patients taking sodium oxybate): Discontinuing sodium oxybate requires management by a neurologist. Alternative treatments for narcolepsy exist (modafinil, armodafinil, solriamfetol, pitolisant) and should be optimised before or at the same time as reducing the dose of sodium oxybate.

# A Final Word.

**THE CENTURION — The system that was outsourcing its stillness has everything it needs to find it again.**

GHB recovery is among the least publicly discussed and least clinically supported recovery journeys that exists. The people who need this guide are often invisible to the systems that would help them: too functional to seek standard treatment, too dependent to manage alone, too stigmatized in some contexts to disclose, and too often told that what they are experiencing is 'not that serious' by people who do not understand the neurobiological reality of GABA-B dependence.

The Recovery Kernel was designed specifically for this group of people—those who are not responding to conventional treatments, not because they lack dedication, but because those treatments are not addressing the unique biological factors in their individual circumstances.

The biology of GHB recovery is clear. The GABA-B receptors upregulate. The glutamate system normalizes. The HPA axis restores. The dopamine system rebuilds. The sleep architecture recovers. The process takes longer than most people are told, and the path through it is narrower than most people are supported through.

But the nervous system that found the stillness it needed in a bottle of GHB is the same nervous system that can find it in a breath, a cold river, a room full of people who understand, a creative practice that absorbs it completely, and a contemplative tradition that has been cultivating this exact internal state for thousands of years.

It built the trap. It can build the door.

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*Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.*

**Crisis: call or text 988 | Emergency: 911 | SAMHSA: 1-800-662-4357**